Exposure to PBDEs in Offices: Connecting Dust, Handwipes and Serum

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Introduction

Polybrominated diphenyl ethers (PBDEs) have been widely used as flame retardants in consumer products and are now ubiquitous in residential indoor air and dust (Stapleton et al. 2005; Allen et al. 2007; Allen et al. 2008). PBDEs can also be measured in human tissues such as serum and breast milk with concentrations increasing over the past few decades (Sjödin et al. 2008; Frederiksen et al. 2009). Sources of exposure include both diet and the residential environment (Stapleton et al. 2005; Wu et al. 2007; Fraser et al. 2009). Associations have been found between PBDE body burdens and house dust (Wu et al. 2007). However, little is known about exposure in the office environment as previous US studies of PBDEs have focused on homes. Many people spend substantial amounts of time in their office where equipment containing PBDEs or other flame-retardants are likely present. In addition, many offices are considered public space, where furniture may be required to meet stricter fire codes. Such flammability standards may increase exposure to PBDEs (Zota et al. 2008). We hypothesize that these factors may increase exposure to PBDEs and other flame retardants in offices. Suspected routes of exposure to PBDEs in the indoor environment include incidental dust ingestion, dermal exposure and inhalation, but the primary exposure pathways are still unclear. Stapleton et al. demonstrated that PBDEs can be detected in handwipes (Stapleton et al. 2008), suggesting that a key exposure route may be incidental ingestion during eating, biting nails, or other hand-to-mouth behaviors. Stapleton et al. estimated exposure via this pathway using exposure factors but were unable to definitively link PBDEs measured on handwipes to body burdens.

The goals of the present study were to examine relationships between PBDE concentrations in the office environment and internal exposure using concurrent measurements of PBDEs in serum, handwipe, and office dust, as well as elucidate pathways by which people working in the office environment are exposed to PBDEs.

Materials and Methods

In the winter of 2009 we collected samples of serum, dust, and handwipes from a convenience sample of 31 participants in the Boston area who spent at least 20 hours a week in an office environment. Blood samples were collected from each participant by a trained phlebotomist. Samples were allowed to coagulate at room temperature for 1-2 hours and then centrifuged for 15 minutes at 1000 x g. Serum aliquots were analyzed for tri- to decaBDEs and lipids at the Centers for Disease Control and

Prevention using established methods (Sjödin et al. 2004).

Dust samples were collected using a cellulose extraction thimble (Whatman International) inserted between the crevice tool and vacuum tube extender of a Eureka Mighty-Mite vacuum cleaner (Allen et al. 2008). Each office was vacuumed for approximately 10 minutes, capturing dust from the surface area of the room including floors under desks and the tops of immovable furniture. Dust samples were sieved to collect particles <500 μm in size. The sieved sample was then placed in a clean amber glass jar and stored at -20°C. Sodium sulfate powder was used as a surrogate for dust in the collection of field blanks. Handwipe samples were collected from each participant in their office environment at least 60 minutes after they had last washed their hands. A sterile gauze pad was immersed in 3 ml of isopropyl alcohol and then used to wipe both the palm and back of hand from wrist to fingertips. Left and right hand samples were analyzed together, providing one measurement per participant. A field blank wipe sample was paired with the collection of each handwipe sample by soaking a gauze pad in isopropyl alcohol and placing it directly into the glass vial.

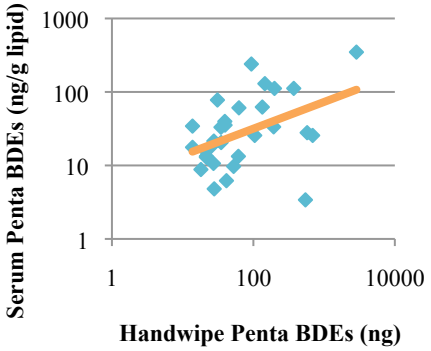
Dust and handwipe samples were analyzed for PBDEs at Duke University using gas chromatography-mass spectrometry operated in electron capture negative ionization mode (GC/ECNI-MS) (Stapleton et al. 2008). Dust samples were blank-corrected using the mean of the appropriate field blanks while handwipes were blank-corrected by using each individual's blank for their sample. For all samples, concentrations below the LOD were substituted with a value of ½ the LOD. All data were natural log-transformed and all statistical analyses were performed using SAS version 9.1.

Results and Discussion

PBDE congeners 28, 47, 99, 100, and 153 were detected in greater than 50% of all serum samples, with the sum of these five congeners (defined here as "pentaBDEs") ranging from 3.4 to 348.4 ng/g lipid with a geometric mean (GM) of 27.2 ng/g lipid. Congeners BDE 17, 66, 85, 154, and 183 were detected in less than 50% of serum samples and were not included in the data analysis. BDE 209 was detected in 20% of serum samples, ranging from <4.8 to 9.7 ng/g lipid. As BDE 209 is the main component of the decaBDE commercial mixture and has somewhat different chemical properties, it was treated separately in data analysis. We report here some of the first concentrations of BDE 209 in serum from the US.

For consistency, we restricted data analysis to the same congeners in dust, handwipe and serum samples. These pentaBDE congeners and BDE 209 were all detected in greater than 50% of both dust and handwipe samples. PentaBDEs in handwipes ranged from 14 to 2845 ng (GM = 69 ng), and BDE 209 ranged from <2 to 105 ng (GM = 12 ng). In office dust, pentaBDE concentrations ranged from 141 to 61264 ng/g (GM = 2167 ng/g), and BDE 209 ranged from 902 to 125,555 ng/g (GM = 4173 ng/g).

Figure 1.
Penta BDEs: Serum vs. Handwipes



PentaBDEs in handwipe and serum samples were correlated, with a Spearman correlation coefficient of 0.44 ($p = 0.01$). See Figure 1. PentaBDEs in handwipes were also divided into low, medium, and high categories based on tertiles. These categories were used in a linear regression model as predictors of pentaBDE serum concentrations (Table 1). Handwipe category was a significant predictor of pentaBDE concentrations in serum ($p = 0.03$), with serum concentrations in the high handwipe category approximately 3.5 times higher than serum concentrations in the low handwipe category, and 2.2 times higher in the medium handwipe category compared to the low handwipe category.

Table 1. PentaBDE Handwipe Tertiles Associated with PentaBDE Concentrations in Serum

HW Penta Category	n	β	Serum Penta GM (ng/g lw)	p-value
Low	10	(ref)	14.00	(ref)
Med	11	2.22	31.15	0.0883
High	10	3.47	48.58	0.0105

PentaBDE concentrations in handwipes were moderately correlated with pentaBDE concentrations in office dust, with a Spearman correlation coefficient of 0.35 ($p = 0.07$). As handwipes were collected in the office environment, a correlation with office dust was expected. PentaBDE concentrations in office dust were only weakly correlated with serum pentaBDEs ($r = 0.17$, $p = 0.38$). Handwipes may be a more biologically relevant measure of dust exposure, possibly integrating across multiple micro-environments. In addition, office workers may not come into contact with all dust in their office (e.g. the floor), only dust that is in their immediate workspace. Handwipes provide a measure of personal exposure to dust, an intermediate step that explains how PBDEs in dust (measured in environment) become PBDEs in people (measure of absorbed dose).

BDE 209 levels in serum, handwipe, and office dust samples followed a similar, albeit weaker, pattern. As BDE 209 was only detected in 20% of serum samples, we analyzed serum BDE 209 as detect vs. non-detect using logistic regression. Using BDE 209 levels in handwipes as a continuous predictor produced an odds ratio (OR) of 1.02 ($p = 0.14$), *i.e.*, a 2% increase in the odds of detecting BDE 209 per unit increase of BDE 209 in handwipes. Use of high vs. low categories of handwipe BDE 209 produced an OR of 2.4 ($p = 0.37$), indicating that people with high levels of BDE 209 on their hands had 2.4 times the odds of having BDE 209 detected in their serum as people with low levels on their hands. These results were not statistically significant, at least partly due to the small percentage of serum samples with detectable BDE 209. Larger studies are needed to further investigate the association between serum BDE 209 and handwipes.

Associations between BDE 209 levels in handwipes and office dust were similar to what we found with pentaBDEs, with a Spearman correlation coefficient of 0.33 ($p = 0.07$). Again, this relationship is not surprising as handwipe samples were collected in the participant's office and may reflect exposure to dust in this environment. The association between BDE 209 concentrations in office dust and detection of BDE 209 in serum was not significant (OR = 1.0, $p = 0.67$). Our ability to examine this association was limited by our small sample size as well as our detection limits for serum BDE 209.

In sum, our research suggests exposure to PBDEs in the work environment may significantly

contribute to PBDE body burden for office workers. Associations between PBDEs found in office dust, handwipes, and serum suggest that potential exposure pathways may involve PBDEs on hands, either through incidental ingestion or dermal absorption.

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